EVALUATION OF CARDIOVASCULAR RISK IN THE ELDERLY: THE FRAMINGHAM STUDY*

WILLIAM B. KANNEL, M.D.

Director
The Framingham Study for the National Institutes of Health
Framingham, Massachusetts

TAVIA GORDON

National Heart, Lung and Blood Institute
Bethesda, Maryland

THE aged are bedeviled by multiple problems which take the joy out of reaching an advanced stage of life. Although disabilities afflicting the aged are many, cardiovascular and cerebrovascular disorders are the most common and account for substantial disability among the aged. Sudden coronary death may be a choice way to depart life at age 80, but slow strangulation by congestive heart failure or the totally incapacitating effects of a stroke are not pleasant rewards for advanced age.

The cohort from the Framingham Study has now entered the geriatric age group, and provides an opportunity to determine whether the cardiovascular risk factors which operate in the aged are the same as those in the young adult. This is important, not as part of a quest for immortality, but rather in hopes of improving the quality of the last years of life. This report examines the utility of standard major risk factors in predicting cardiovascular disease in the elderly. Implications for the prevention of cardiovascular disease in the aged will be considered.

The Framingham Heart Disease Epidemiology Study, which forms the basis for this report, was initiated in 1949 to explore the epidemiology of cardiovascular disease in a general population sample of 5,209 men and women aged 30 to 62 years on entry. The study has been in continuous operation since that time, examining subjects for the initial development

*Presented as part of a *Symposium on Geriatric Medicine* held by the Section on Geriatric Medicine of the New York Academy of Medicine and the American Geriatric Society of the Academy June 8, 1977.

of cardiovascular disease biennially. Detailed descriptions of the sampling procedure, response rate, methods of examination, laboratory procedures, and the criteria for disease outcomes have been reported in detail elsewhere.[1] The town of Framingham is located in Massachusetts, 25 miles west of Boston. The sample was representative of the general population, although the response to invitation to participate was only 68%. Follow-up has been reasonably complete, with only 2% lost.

The number of participants which forms the basis for Tables II through VIII ranged from 500 to 940 in each 10-year age group. The tables included are based on data from 20 years of follow-up. The incidence tables deal with incidence between one biennial examination and the next. Persons were characterized anew at each biennial examination and shifted from one age group to the next as they grew older. The sum of the experience in the 10 intervals of follow-up was expressed as an average annual incidence rate. Thus, persons frequently contribute to two different age groups, allowing an expansion of the age-specific experience. Because a person may contribute from two to 10 person years experience in an age group, precise counts of persons on which the tables are based cannot be given.

Causes of Mortality in the Aged

Diseases of the heart and blood vessels are the most important cause of death in old age as well as in younger adults. More than half of all mortality beyond age 65 is due to cardiac and cerebral vascular disease. As in the younger adult, coronary heart disease is a major cause of this cardiovascular mortality.

Cardiovascular disease outranks any other cause of mortality in persons beyond age 65, followed by cancer and stroke.[1] If deaths from cardiovascular and renal diseases were eliminated in the United States, the National Center of Health Statistics estimates that life expectancy at birth would gain almost 11 years (see Table I). Even at age 65, eliminating deaths from cardiovascular and renal diseases should yield a 10-year gain in lifespan. If deaths from heart disease alone were eliminated, the corresponding figures would be 5.9 and 4.9 years, respectively (Table I).

Cardiovascular Morbidity

As with mortality, morbidity from cardiac, cerebral, and peripheral vascular disease is considerable beyond age 65 (Table II). Clinical exami-

TABLE I. ESTIMATED GAIN IN LIFE EXPECTANCY AT BIRTH AND AT AGE 65 WITH THE ELIMINATION OF VARIOUS CAUSES OF DEATH*

Cause of death	*Gain in expectation of life if cause was eliminated (yr.)*	
	At birth	*At age 65*
Major cardiovascular and renal diseases	10.9	10.0
Heart diseases	5.9	4.9
Vascular diseases affecting the central nervous system	1.3	1.2
Malignant neoplasms	2.3	1.2
Accidents, excluding those caused by motor vehicles	0.6	0.1
Motor-vehicle accidents	0.6	0.1
Influenza and pneumonia	0.5	0.2
Infectious diseases (excluding tuberculosis)	0.2	0.1
Diabetes mellitus	0.2	0.2
Tuberculosis	0.1	0.0

*Source: Life tables published by the National Center of Health Statistics, U.S. Public Health Service and U.S. Bureau of Census: *Some Demographic Aspects of Aging in the United States*, Washington, D.C., Govt. Print. Off., 1973.

nations of a variety of populations indicate a high prevalence of heart disease in those more than 65 years of age.[2] In this age group the prevalence of coronary heart disease appears to be about 20% in men and 12% to 66% in women, rheumatic heart disease in the order of 2% to 3%, and aortic stenosis about 4%. Pulmonary heart disease is less common and is virtually confined to men. In addition, definite and unclassifiable heart disease is found in some 4% to 8% of those 65 to 74 years of age and in 8% to 10% of those more than 75 years of age. Thus, clinical studies indicate a very high prevalence of cardiac disease in otherwise fit old people. About 40% are afflicted in the 65-to-74-year age group and 50% or more of those beyond age 75.[2] Objective corroborative evidence of this high prevalence is provided by surveys of electrocardiographic abnormalities which reveal aberrations in 40% to 60% of old people.[3]

Information on the *incidence* of heart disease in old age is scarce, but it is clear from the Framingham study that the incidence of all major cardiac and vascular disorders is substantial (Table II). The advantage women have over men in this area wanes progressively with advancing age.

A study by Akhtar et al. of 299 randomly selected old people whose disability made them unable to live at home without help showed that 20% had clinical evidence of heart disease which was evaluated as at least

TABLE II. AVERAGE ANNUAL INCIDENCE* OF CARDIOVASCULAR EVENTS IN RELATION TO AGE AND SEX AMONG MEN AND WOMEN 45 TO 74 YEARS OF AGE WHO PARTICIPATED IN THE FRAMINGHAM STUDY: 20-YEAR FOLLOW-UP

Age (Yrs.)	*Coronary heart disease*		*Cerebro vascular accident*		*Peripheral arterial disease*		*Congestive heart failure*	
	Men	*Women*	*Men*	*Women*	*Men*	*Women*	*Men*	*Women*
45 to 54	9.9	3.1	2.0	0.9	1.8	0.6	1.8	0.8
55 to 64	20.8	9.5	3.2	2.9	5.1	1.9	4.3	2.7
65 to 74	20.4	14.5	8.4	8.6	6.3	3.8	8.2	6.8

*Per 1,000 population

contributory.[4] Among the elderly, heart disease substantially impedes independent existence in the community.

Aging and Cardiovascular Disease

It is difficult to specify precisely what aging of the cardiovascular system is. As people get older it is clear that they have had longer exposure to noxious influences and that the vascular preconditions of clinically evident disease are likely to be more advanced than in the young. With progressive closing down of circulation due to atherothrombotic, embolic, and arteriolar disease, a decline in organ function is likely with advancing age.

Most findings in the heart and blood vessels regarded as typical of the elderly are not inevitable expressions of normal senescence but rather of disease, which increases with age. Coronary arteries almost free of atheroma, normal valves, and a normal sized heart are not rarities, even among people 90 years old.[2]

Clearly, the increased incidence of cardiovascular disease with age is not solely a consequence of the rising level of identified cardiovascular risk factors as people grow older. An examination of the gradient of risk in the Framingham cohort reveals a steep rise with age among even those whose characteristics place them at low risk (Table III). However, multiple risk factors can make one old beyond one's years with respect to the cardiovascular apparatus. Among the aged as well as among the young, risk is greatly augmented by the presence of multiple risk factors such as those shown in Table IV.

TABLE III. AGE TREND IN EIGHT-YEAR PROBABILITY OF CARDIOVASCULAR DISEASE (PERCENT) AMONG MEN AND WOMEN 35 to 70 YEARS OF AGE WHO PARTICIPATED IN THE FRAMINGHAM STUDY: 18-YEAR FOLLOW-UP*

Age (yrs.)	*Optimal risk category†*		*Poor risk category‡*	
	Men	*Women*	*Men*	*Women*
35	0.6	0.4	60.2	19.5
40	1.2	0.7	70.8	28.4
45	2.2	1.3	77.8	38.0
50	3.7	2.2	81.9	47.0
55	5.5	3.4	84.1	54.7
60	7.4	5.1	84.8	60.6
65	9.0	7.0	84.0	64.9
70	10.0	9.0	81.7	67.5

*Based on smoothed data.
†Systolic blood pressure: 105 mm. Hg, serum cholesterol level: 185 mg./dl, no reaction to the glucose intolerance test, does not smoke cigarettes, and has no electrocardiographic evidence of left-ventricular hypertrophy.
‡Systolic blood pressure: 195 mm. Hg, serum cholesterol level: 335, mg./dl., positive reaction to the glucose intolerance test, smokes cigarettes, or has electrocardiographic evidence of left-ventricular hypertrophy.

To forestall cardiovascular disasters in the aged, people who are especially vulnerable to cardiovascular disease must be treated long before symptoms appear. To identify such people, a cardiovascular risk profile should be obtained as a regular part of the physician's office examination to assess the magnitude of the risk and so that multifactorial preventive measures can be instituted as soon as indicated. Ideally, this should be done as early in life as possible.

Cardiovascular Profiles in the Aged

It has been shown that the risk of cardiovascular events in young and middle-aged people can be estimated efficiently by synthesizing a number of risk factors into a composite function score.[5] This is more useful than a categorical assessment which arbitrarily designates abnormal or unacceptable levels for each of the major risk factors because it also detects people at high risk from a combination of marginal abnormalities.

Total serum cholesterol levels and the amount of cigarette smoking are useful in predicting the risk of coronary heart disease in those less than age 65, but appear less useful in advanced age. Nevertheless, a risk-function score which includes these characteristics along with blood pressure, glucose intolerance, and ECG-LVH remains an effective predictor of cardiovascular risk for those 65 to 74 years of age. With this set of variables it is possible to identify a tenth of the population of men in this age group from

TABLE IV. PERCENTAGE OF CASES OF CORONARY DISEASE, BRAIN INFARCTION, AND INTERMITTENT CLAUDICATION IN THE UPPER DECILE OF MULTIVARIATE RISK* AMONG MEN AND WOMEN 45 TO 74 YEARS OF AGE WHO PARTICIPATED IN THE FRAMINGHAM STUDY: 16-YEAR FOLLOW-UP

Age (yrs.)	*Coronary disease*		*Brain infarction*		*Intermittent claudication*	
	Men	*Women*	*Men*	*Women*	*Men*	*Women*
45 to 54	25.9	20.0	54.5	44.4	30.0	60.0
55 to 64	26.7	25.7	52.3	42.9	46.7	42.9
65 to 74	21.3	40.9	57.1	45.5	26.7	50.0

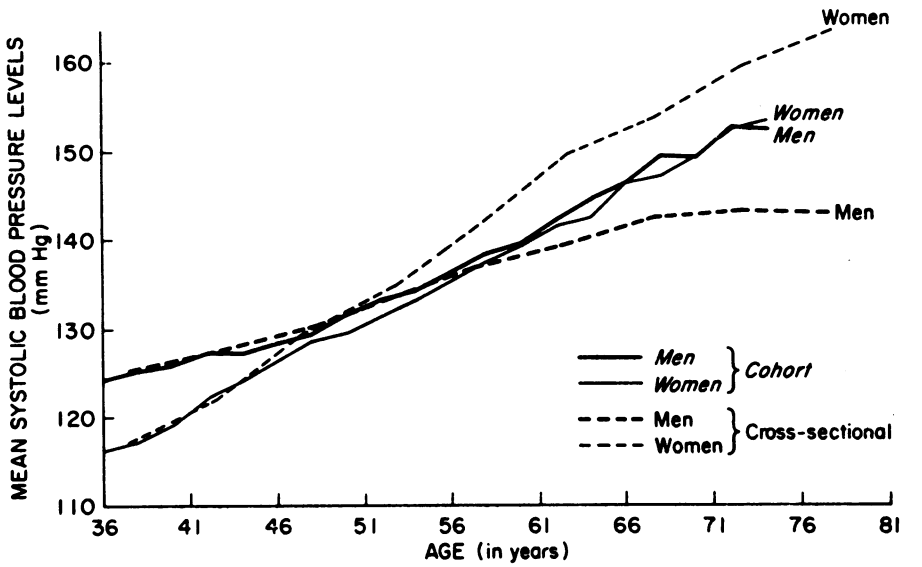
*Based upon systolic blood pressure, serum cholesterol, number of cigarettes smoked, electrocardiographic evidence of left-ventricular hypertrophy, and glucose tolerance.

which about 21% of coronary heart disease, 57% of cerebral infarction, and 28% of intermittent claudication will arise (Table IV). Corresponding figures for women in the upper decile of multivariate risk are about 41%, 45%, and 50%—even more impressive than for men.

Because two components of this risk profile are of minimal use beyond age 65, more potent constituents have been sought. In the older ages the total serum cholesterol level has little predictive value for men. However, analysis of the lipoprotein components of the total cholesterol has revealed two elements associated with coronary heart disease—HDL-cholesterol, which is protective, and LDL-cholesterol, which promotes coronary heart disease.[6] In addition, serum triglyceride levels, systolic blood pressure, relative weight, diabetes mellitus, and ECG-LVH taken alone were each found to be associated with coronary heart disease in the elderly. However, when the contribution of each was examined taking the other variables into account, obesity and elevated serum triglyceride levels contribute a little to risk. Using the remaining variables together, it was possible to estimate the risk of coronary artery disease in people 50 to 80 years old accurately enough so that 30% of the cases occur in the 10% of the population at highest risk. This prediction is as true for people 70 to 79 years old as for those in their 50s.[7]

Hypertension in the Elderly

Because blood pressure stands out as the major risk factor in the elderly, a detailed examination of its contribution to cardiovascular risk in the aged is warranted. Hypertension is the most readily and objectively identified



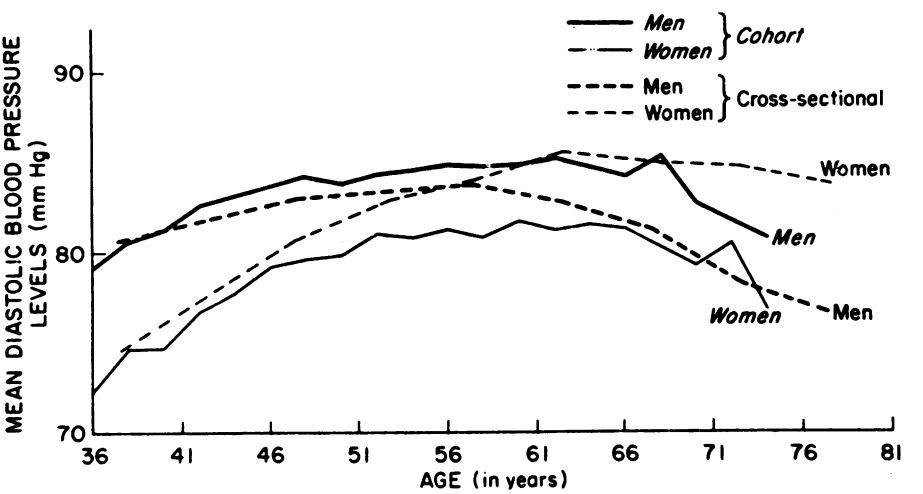


Fig. 1. Average age trends in blood-pressure levels for men and women, based upon cross-sectional and cohort data on participants in the Framingham study. Data was obtained at biennial examinations No. 3 to 10.

major risk factor promoting cardiovascular disease in people of all ages. It is, theoretically, an easily detected and easily controlled contributor to cardiovascular disease. A variety of antihypertensive agents, with tolerable side effects if used skillfully, can normalize most elevated blood pressures among the elderly. The evidence is convincing that when this is done—even in people of middle age and older who have sustained diastolic hypertension and even after the appearance of target-organ involvement—cardiovascular morbidity and mortality are substantially reduced after a relatively short period of treatment.[8] Cardiovascular disease is more prominent as a cause of death and disability among aged hypertensives than young. We need detailed information on the role of blood pressure, the factors that affect its impact, and the indications for treatment among the aged.

Age Trends in Blood Pressure

From prevalence data it seems clear that blood pressure increases with age in most population samples. That it fails to do so in some primitive, isolated cultures suggests that this is neither inevitable nor a physiologic consequence of aging, [9,10] but that it does rise in most populations suggests that these elevations may be difficult to avoid.

Evaluation of age trends in blood pressure reveals a disproportionate rise in systolic blood pressure with advancing age compared to diastolic pressure. Cross-sectional data show that both systolic and diastolic pressures increase with age in adult life, and systolic pressures continue to rise into the seventh decade in men (Figure 1). Diastolic pressures seem to peak sooner and, in men, to decline precipitously after age 56. In women, pressures initially are lower than in men of the same age, and rise more steeply with age until about age 50, when they equal those of men, and then progressively increase to exceed those in men. In cross-sectional data this cross-over in pressures is observed for both the systolic and diastolic components.

Longitudinal data, reflecting changing pressure as the Framingham cohort ages, reveal, instead, diastolic pressures that are essentially parallel in the sexes, with women's pressure persistently lower than men's (Figure 1). These cohort data also show that systolic pressures of women initially are lower than those of men, but converge on those of men by age 60 but never exceed them. Data indicate a disproportionate rise in systolic pressure with advancing age relative to diastolic pressure, presumably from progressive

TABLE V. RISK OF CARDIOVASCULAR DISEASE* IN RELATION TO HYPERTENSIVE STATUS AND AGE, BASED UPON MEN AND WOMEN 45 TO 74 YEARS OF AGE WHO PARTICIPATED IN THE FRAMINGHAM STUDY: 20-YEAR FOLLOW-UP

Hypertensive status	*Men*			*Women*		
	45 to 54 yrs.	*55 to 64 yrs.*	*65 to 74 yrs.*	*45 to 54 yrs.*	*55 to 64 yrs.*	*65 to 74 yrs.*
Normal	8.6	15.6	17.1	2.7	6.1	8.6
Borderline	14.5	30.4	32.7	6.1	14.4	22.5
Hypertension	23.6	43.9	51.0	9.7	23.7	35.6

*Incidence per 1,000 population
Normal = <140/90 mm. Hg, borderline = 140–160/90–95 mm. Hg, hypertensive = < 160/95 mm. Hg

loss of arterial elasticity. The reason for the difference in blood pressure and age trends obtained cross-sectionally and longitudinally in the same cohort is not clear. Because blood pressures increase with age, some contend that this is either a physiological sign of progressive increase in arterial rigidity or a compensatory change to insure perfusion through narrowed vessels. However, whether or not the rise in blood pressure with age is inevitable, it clearly is detrimental to cardiovascular health. Neither absolute nor relative risk is lower in elderly hypertensives than in younger groups (Table V). Even for systolic blood pressure, gradients of risk of cardiovascular sequelae do not indicate waning impact with advancing age. Further, there is no evidence that cardiovascular sequelae in the elderly are more closely related to the diastolic than to the systolic component of the blood pressure.[11] Thus, there is no indication that the disproportionate rise in systolic pressure with age is innocuous and, even at low diastolic pressure, risk depends on the accompanying systolic pressure.[11]

It could be argued that the high risk associated with elevated systolic pressure in the aged derives not from the pressure but from vascular rigidity, of which systolic hypertension is only a sign.[12] If this were so, one would expect a diminishing effect of systolic pressure with advancing age; this does not occur. Moreover, a wide pulse pressure (the hallmark of vascular rigidity) is not more strongly associated with stroke than with systolic pressure and its impact does not increase with advancing age.[11] When systolic pressure is taken into account, pulse pressure contributes to risk at any age.[11]

There is also no evidence that, as some contend, elderly women tolerate hypertension better than men. The attributable risk of cardiovascular mor-

TABLE VI. ATTRIBUTABLE RISK* OF HYPERTENSION IN RELATION TO AGE, BASED UPON MEN AND WOMEN 45 TO 74 YEARS OF AGE WHO PARTICIPATED IN THE FRAMINGHAM STUDY: 20-YEAR FOLLOW-UP

Age (Yrs.)	*Overall mortality*	*Cardiovascular mortality*	*Cardiovascular morbidity*
		Men	
45 to 54	17.9	29.3	16.4
55 to 64	16.1	21.4	17.9
65 to 74	8.4	12.9	18.8
		Women	
45 to 54	12.0	28.6	17.4
55 to 64	8.5	17.5	26.5
65 to 74	14.9	34.5	26.9

*Computed by subtracting the rate for nonhypertensives from the rate for the total population and dividing this quantity by the rate for the total population and expressed as a percentage.

TABLE VII. PERCENTAGE OF HYPERTENSIVES DEVELOPING CARDIOVASCULAR DISEASE WITHOUT PRIOR EVIDENCE OF TARGET-ORGAN INVOLVEMENT, BASED UPON MEN AND WOMEN 35 TO 74 YEARS OF AGE WHO PARTICIPATED IN THE FRAMINGHAM STUDY: 20-YEAR FOLLOW-UP

Age	*Men*	*Women*
35 to 44	75%	33%
45 to 54	58%	56%
55 to 64	48%	38%
65 to 74	33%	33%
All Ages	50%	39%

*Roentgenographic evidence of cardiac enlargement; electrocardiographic abnormalities such as left-ventricular hypertrophy, intraventricular block, or nonspecific abnormalities; or albuminuria.

bidity and mortality actually is greater for women more than 65 years old than for men of the same age (Table VI), and for women, unlike men, there is no reduction in attributable risk with advancing age.

It is unsafe to await the appearance of target-organ involvement in elderly hypertensives before treating them, because about half the cardiovascular sequelae in men and 40% in women appear before such evidence can be detected on biennial examination (Table VII). Nor is it safe to ignore labile hypertension. Labile blood-pressure elevations are considered less serious than fixed ones, a logical fallacy, because all blood pressures are labile, high pressures considerably more so than low ones.[14] It is also unsafe to judge the need for treatment by the lowest blood pressure

TABLE VIII. PERCENTAGE OF PEOPLE WITH VARIOUS SYSTOLIC BLOOD PRESSURES* WHO RECEIVED ANTIHYPERTENSIVE DRUGS. BASED UPON MEN AND WOMEN 35 TO 74 YEARS OF AGE WHO PARTICIPATED IN THE FRAMINGHAM STUDY

Systolic blood pressure (mm.Hg)	*Men*			
	35 to 44 yrs.	*45 to 54 yrs.*	*55 to 64 yrs.*	*65 to 74 yrs.*
140 to 149	2.26	2.23	2.49	1.66
150 to 159	7.75	5.74	7.12	7.02
160 to 169	10.42	8.25	8.55	7.02
170 to 179	12.50	18.89	15.44	14.81
180 to 189	23.08	27.27	23.75	14.29
190 to 199	25.00	26.92	30.61	17.39
200 or more	40.00	27.78	36.73	20.83
Systolic blood pressure (mm.Hg)	*Women*			
	35 to 44 yrs.	*45 to 54 yrs.*	*55 to 64 yrs.*	*65 to 74 yrs.*
140 to 149	3.45	5.30	5.86	5.95
150 to 159	10.53	10.84	6.35	7.97
160 to 169	10.71	14.76	11.60	9.47
170 to 179	22.22	20.77	16.28	16.13
180 to 189	22.22	31.43	17.45	20.25
190 to 199	40.00	33.33	40.85	26.92
200 or more	42.85	53.33	39.58	32.26

* On examination preceding the first use of antihypertensive drugs. Based upon examinations 4 through 10.

recorded on an elderly patient if the average pressure is high.

Even in the Framingham cohort, where hypertension is routinely detected and reported biennially to the patient and his physician, a sizeable number of people with elevated blood pressures get no treatment, even for severe hypertension, and the portion treated drops sharply after age 65 (Table VIII). Clearly, physicians are less inclined to treat elderly hypertensives, which is unfortunate because hypertension is the most potent, most common, and most remediable contributor to cardiovascular disease in the elderly. It is also tragic, in view of evidence that life can be prolonged and strokes and heart failure can be deferred by treatment.[8] This therapeutic neglect seems to come not only from difficulty in getting this age group to follow treatment but from the medical misconceptions enumerated above.

Other Risk Factors

Little evidence supports intervention on other risk factors suggested as

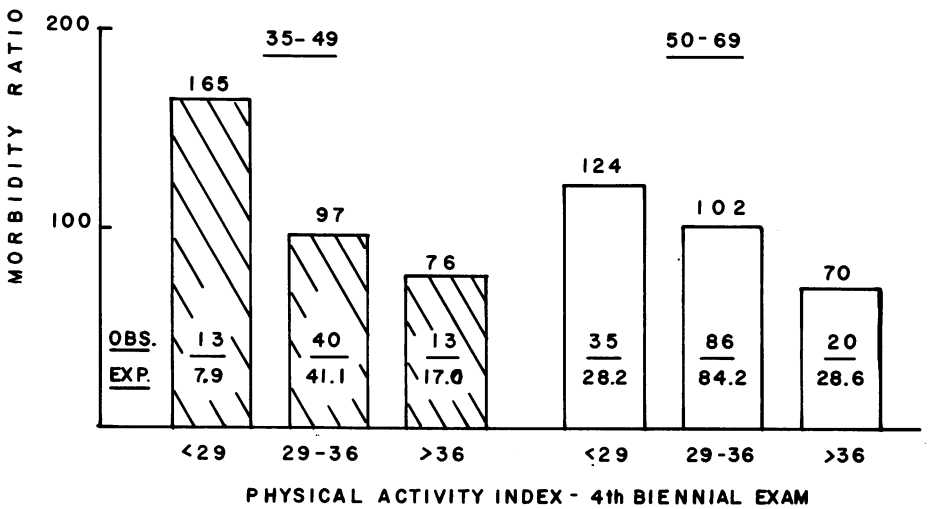


Fig. 2. Risk of coronary heart disease (10 years) in relation to age and physical activity status, based upon men 35 to 69 years of age participating in the Framingham study. Data was obtained at the fourth biennial examination. OBS. = observed, EXP. = expected. Reproduced by permission from Kannel, W. B.: Some lessons in cardiovascular epidemiology from Framingham. *Am. J. Cardiol. 37:* 279, 1976; Kannel, W. B.: Prevention of heart disease in the young coronary candidate. *Primary Care 4:* 241, 1977.

promoting cardiovascular disease. Some preventive measures, however, seem worthwhile for other reasons and should be encouraged.

Physical Activity

Findings incriminating lack of physical activity in the development of cardiovascular disease are inconsistent.[14-17] It is not yet clear whether vigorous leisure or work activity, fitness, or high-intensity endurance exercise are required. Although regular physical activity seems desirable for the elderly, it is hard to find clear evidence that this is associated with reduced morbidity and mortality or that the effect would be substantial were blood pressure controlled or already normal. Framingham data indicated some apparent benefit from exercise beyond age 50, and the levels of activity noted seem rather modest (Figure 2). In any event, it is difficult and perhaps unwise to motivate the elderly either to undertake vigorous regular exercise or to enjoy it, but such moderate exercise as walking and climbing stairs may be beneficial.

Overweight

Prospective data show little effect from overweight on cardiovascular

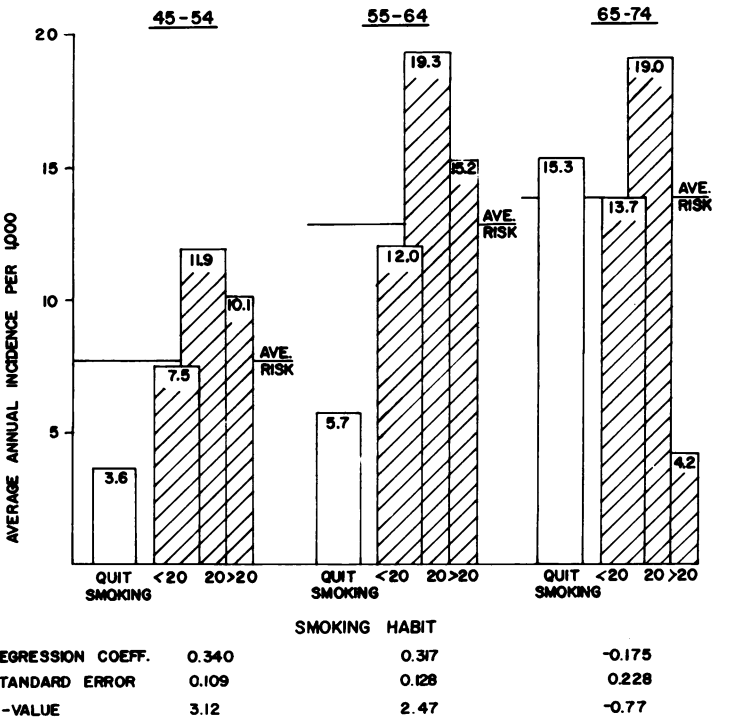


Fig. 3. Incidence of coronary attacks among cigarette smokers in relation to subsequent cigarette habit, based upon examination of men 45 to 74 years of age who participated in the Framingham study: 18-year follow-up. AVE. = average, COEFF. = coefficient. Reproduced by permission from Kannel, W. B.: Some lessons in cardiovasular epidemiology from Framingham. *Am. J. Cardiol. 37:* 280, 1976; Kannel, W. B.: Prospects for prevention of atherosclerosis in the young. *Aust. N.Z. J. Med. 6:* 417, 1976; Kannel, W. B.: Recent highlights from the Framingham study. *Aust. N.Z. J. Med.6:* 381, 1976; Kannel, W. B.: Preventive cardiology: What should the clinician be doing about it? *Postgrad. Med. 61:* 80, 1977, Kannel, W. B.: Prevention of heart disease in the young coronary candidate. *Primary Care 4:* 235, 1977.

morbidity and mortality in the aged. Where there is some effect, it is greatly reduced if other factors which generally accompany obesity are considered. Nevertheless, because weight changes are accompanied by changes in a variety of atherogenic traits, slimming appears to have a sound rationale. Weight reduction also should reduce the cardiac work load and improve exercise tolerance in those with already compromised circulation.

Cigarettes

Some large-scale prospective studies show a slightly higher coronary mortality among cigarette smokers, even at age 75 to 84, but these studies

also have shown that the relative risk attached to smoking diminishes with advancing age.[18-20] Evidence of the benefit of quitting cigarettes is provided by prospective studies which document a reduced incidence of coronary attacks in those who gave up smoking,[21-23] but this effect cannot be demonstrated after age 65 (Figure 3). The overall death rate is considerably lower in those who give up smoking, despite the lack of change in the rate of new coronary attacks.[21] There is adequate reason to advise the elderly to give up cigarettes, whether or not this reduces the risk of coronary attacks.

Lipids

Elevations of LDL-cholesterol in the elderly, as at younger ages, are associated with increased risk of coronary heart disease (CHD), while HDL elevations are associated with decreased risk of CHD. There is no convincing evidence, however, that lowering LDL or raising HDL levels decreases risk, and in the elderly there is the additional presumption that atherosclerotic effects may be so far advanced that altering lipids is unlikely to have prophylactic value. Dietary control could be defended on grounds that some who do not have advanced disease might benefit.

Diabetes Mellitus

Patients with diabetes mellitus appear to have a poor myocardium and a high risk of congestive heart failure.[24] Women fare worse than men when they develop diabetes, and lose their advantage over men with respect to cardiovascular mortality. There is little evidence that control of hyperglycemia, either by oral hypoglycemic agents or insulin, improves cardiovascular mortality.[25]

Other Factors

Neither coffee nor moderate alcohol use seem to be associated with cardiovascular risk.[26] Reduction of emotional stress would seem reasonable, but there is little convincing evidence to support the recommendation, nor does available evidence encourage prophylactic use of aspirin or beta-adrenergic blocking agents. Treatment of hyperuricemia or subclinical hypothyroidism to reduce cardiovascular risk also lacks a factual basis.

Discussion

Logically, preventive measures should start early in life to reduce

TABLE IX. PERCENTAGE DECLINE IN AGE-SPECIFIC MORTALITY FROM CORONARY HEART DISEASE AND STROKE, 1968 TO 1975.

Age (yrs.)	*Both sexes*		*White males*	
	CHD	*CVA*	*CHD*	*CVA*
35 to 44	24.7	29.1	23.6	27.5
45 to 54	18.4	27.8	17.7	27.6
55 to 64	18.7	24.7	17.6	23.1
65 to 74	20.4	26.0	17.8	24.2
75 to 84	15.2	18.3	11.8	17.0
85 or more	21.7	26.4	18.3	25.3

CHD = coronary heart disease. CVA = cerebral vascular accident

cardiovascular diseases in both the elderly and the young. This does not necessarily mean that all preventive measures are worthless in advanced age, and the opposite may be true because of high cardiovascular mortality in the elderly. The absolute short-term effect of preventive measures actually may be greater in the elderly than in the young. Because life expectancy is short and freedom from cardiovascular disasters could enhance the quality if not the number of remaining years of life for the elderly, control of risk factors seems justified.

There is some indication that cardiovascular mortality is not an inevitable liability of aging. A declining trend in cardiovascular and cerebrovascular mortality in both elderly and young adults has been noted (Table IX). Although this does not eliminate the need for controlled preventive trials involving the aged, it does justify some optimism about the efficacy of control of cardiovascular disease in the aged.

Intervention programs to modify risk attributes among the elderly face a formidable problem because of the high prevalence of the contributors to cardiovascular disease in this age group (Table X). Even confining attention to hypertension, we must contend with anywhere from 27% to 48% of the aged population. In addition, effective control of cardiovascular risk factors in this group requires a change in the emphasis and attitudes of physicians and other health workers. Old age is not a sin, and the aged deserve preventive management as well as the young.

The major risk factors, even taken together, do not entirely explain the variance in incidence either within or between populations, nor different incidences between the sexes. It is possible that other major risk attributes exist among the aged, yet to be identified. The dissociation of the total serum cholesterol into the two components, one promoting and the other

TABLE X. PERCENTAGE PREVALENCE OF SELECTED RISK FACTORS IN THE UNITED STATES, BASED UPON VARIOUS YEARS FROM 1948 TO 1970

Age (yrs.)	*Inactivity*	*Obesity*	*Hyper-tension*	*Cigarette smoking*	*Diabetes mellitus*	*Hypercholes-terolemia*	*ECG-LVH**
				Men			
35 to 44	12.1	12.5	13.5	48.6	1.1	20.2	2.9
45 to 54	16.9	14.7	18.3	43.1	1.1	25.7	4.8
55 to 64	21.0	12.5	22.3	37.4	3.3	23.5	10.1
65 to 74	27.1	12.7	27.1	22.8†	3.2	21.6	7.1
				Women			
35 to 44	13.3	20.1	8.5	38.8	0.8	12.9	0.9
45 to 54	19.3	24.2	18.2	36.1	2.9	28.0	3.6
55 to 64	30.8	30.9	31.2	24.2	3.2	49.7	4.1
65 to 74	39.0	27.2	47.6	10.2†	6.1	51.0	9.6

*Data for Framingham, Mass. only

†65 years and older

Inactivity = average oxygen consumption of less than 0.30 liter/min. (data given for 1954 to 1958); obesity = weight of 20% or more above median (data given for 1960 to 1962); hypertension = blood pressure of at least 160/90 mm. Hg (data given for 1960 to 1962); cigarette smoking = existing habit (data given for 1970); diabetes mellitus = medically treated disease (data given for 1960 to 1962); hypercholesterolemia = serum cholesterol of at least 260 mg.% /100 mg. (data given for 1960 to 1962); ECG-LVH = electrocardiographic evidence of left-ventricular hypertrophy (data given for 1948-1953).

protecting against cardiovascular disease, is an example of how continued search for additional risk factors may be fruitful. However, we must appreciate that the factors which already have been delineated do identify high-risk subgroups of the elderly population for preventive management. Because determinants of the high-level risk factors which contribute to cardiovascular disease in the elderly are understood poorly, treatment must be confined to their correction.

Health agencies would seem well advised to promote the cardiovascular profile concept and to seek proof of the efficacy of advocated preventive measures in the elderly. Better techniques for modifying behavior in the elderly to correct risk factors require development. While most physicians and patients would prefer medicine to counteract a lifetime of faulty living habits, an acceptable pharmacologic solution to cardiovascular disease in this age group seems unlikely. Even control of hypertension will require mobilization of extensive community resources. Ultimately, we must learn about the causes of risk factors in the aged population and either prevent the appearance of these causes or correct them as soon as they appear.

Summary

Cardiovascular disease, the major cause of disability and death in the aged, is not an inevitable result of senescence, but is related to identified risk factors. Using a set of risk attributes, each of which exerts an independent effect in advanced age, it is possible to estimate risk over a wide range and thus to identify the 10% of the elderly who will account for about a third of future coronary artery disease and an even larger proportion of strokes and congestive heart failure. High-density lipoprotein (HDL)-cholesterol, low-density lipoprotein (LDL)-cholesterol, systolic blood pressure, diabetes, and electrocardiographic evidence of left-ventricular hypertrophy (ECG-LVH) are all, even when considered jointly, associated with the risk of developing coronary heart disease in the aged.

Hypertension is the key remediable contributor to cardiovascular morbidity and mortality in the elderly, yet its prophylactic treatment is grossly deficient. This is probably because of patients' difficulties in adhering to recommended treatment and because of misconceptions which many physicians seem to have about the role of hypertension in promoting cardiovascular disease in the elderly.

Ability to predict cardiovascular disease in the elderly from identified risk factors is no guarantee that intervention to eliminate these factors necessarily will be fruitful. However, because risk does vary widely in relation to identified factors, it would seem worthwhile to determine whether control of the risk factors which operate in advanced age can improve the quality of life among the aged.

REFERENCES

1. National Center for Health Statistics, U.S. Public Health Service, and U.S. Bureau of Census: *Some Demographic Aspects of Aging in the United States.* Washington, D.C., Govt. Print. Off., 1973.
2. Caird, F. I., and Kennedy, R. D.: Epidemiology of Heart Disease in Old Age. In: *Cardiology in Old Age,* Caird, F. I., Dall, J. L. C., Kennedy, R. D., editors. New York and London, Plenum, 1976. pp. 1-10.
3. Campbell, A., Caird, F. I., and Jackson, T. R.: Prevalence of abnormalities of electrocardiogram in old people. *Brit. Heart J. 36*:1005-11, 1974.
4. Akhtar, A. J., Broe, G. A., Crombie, A., McLean, W. M. R., et. al.: Disability and dependence in the elderly at home. *Age Ageing 2*:102-11, 1973.
5. Kannel, W. B., McGee, D., and Gordon, T.: A general cardiovascular risk profile: The Framingham study. *Am. J Cardiol 38*:46-51, 1976.
6. Gordon, T. Castelli, W. P., Hjortland, M. C., Kannel, W. B., and Dawber, T. R.: High density lipoprotein as a protective factor against coronary heart disease: The Framingham study. *Am. J. Med. 62*:707-14, 1977.
7. Gordon, T., Castelli, W. P., Hjortland, M. C., et. al.: Predicting coronary heart disease in middle-aged and older persons. *J.A.M.A. 238*:497-99, 1977.
8. Freis, E. D.: Medical treatment of chronic hypertension. *Mod. Concepts Cardiov. Dis. 40*:17-22, 1971.
9. Page, L. B. and Sidd, J. J.: Medical management of primary hypertension. 2. *N.Engl. J. Med. 287*:1018-23, 1972.
10. Maddocks, I.: Possible absence of essential hypertension in two complete Pacific Island populations. *Lancet 2*:396-99, 1961.
11. Kannel, W. B., Dawber, T. R., Sorlie, P., et. al.: Components of blood pressure and risk of atherothrombotic brain infarction: The Framingham study. *Stroke 7*:327-31, 1976.
12. Dawber, T. R., Thomas, H. E., Jr., and McNamara, P. M.: Characteristics of the dicrotic notch of the arterial pulse wave in coronary heart disease. *Angiology 24*:244-55, 1973.
13. Gordon, T., Sorlie, P., and Kannel, W. B.: Problems in the assessment of blood pressure: The Framingham study. *Int. J. Epidemiol. 5*:327-34, 1976.
14. Hammond, E. C. and Garfinkel, L.: Coronary heart disease, stroke and aortic aneurysm. Factors in the etiology. *Arch. Environ. Health. 19*:167-82, 1969.
15. Morris, J. N., Chave, S. P., Adam, C., et al.: Vigorous exercise in leisure time and the incidence of coronary heart disease. *Lancet 1*:333-39, 1973.
16. Frank, C. W., Weinblatt, E., Shapiro, S., et al.: Physical inactivity as a lethal factor in myocardial infarction among men. *Circulation 34*:1022-33, 1966.
17. Letunov, S. P. and Motylyanskaya, R. E.: Preventive Significance of Exercise Training in Elderly Adults. In: *Prevention of Ischemic Heart Disease: Principles and Practice,* Raab, W., editor. Springfield, Thomas, 1966, pp. 316-20.
18. Hammond, E. C.: Smoking in relation to death rates of one million men and women. *Nat. Cancer Inst. Monogr. 19*:127-204, 1966.
19. Kahn, H. A.: The Dorn study of smoking and mortality among U.S. veterans: Report on eight and one-half years of observation. *Nat. Cancer Inst. Monogr.*

19:1-125, 1966.

20. Doll, R. and Hill, A. B.: Mortality in relation to smoking: Ten years' observations of British doctors, *Brit. Med. J. 1*:1399-1410, 1460-67, 1964.
21. Gordon, T., Kannel, W. B., and McGee, D.: Death and coronary attacks in men after giving up cigarette smoking. *Lancet* 2:1345-48, 1974.
22. Stamler, J. et al.: Risk Factors: Their Role in the Etiology and Pathogenesis of the Atherosclerotic Disease. In: *The Pathogenesis of Atherosclerosis*, Wissler, R. and Greer, J. C., editors. Baltimore, Williams and Wilkins, 1972, pp. 41-119.
23. Doll, R., and Pike, M. C.: Trends in mortality among British doctors in relation to their smoking habits. *J. Roy Coll. Phys.* (London) *6*:216-22, 1972.
24. Kannel, W. B., Hjortland, M., and Castelli, W. P.: Role of diabetes in congestive heart failure: The Framingham study. *Am. J. Cardiol. 34*:29-34, 1974.
25. Meinert, C. L., Knatterud, G. L., Prout, T. E., and Klimt, C. R.: A study of the effects of hypoglycemia agents on vascular complications in patients with adult-onset diabetes. II. Mortality results. *Diabetes* (Suppl.) *19*:789-830, 1970.
26. Dawber, T. R., Kannel, W. B., and Gordon, T.: Coffee and cardiovascular disease: Observations from the Framingham study. *N. Engl. J. Med. 291*:871-874, 1974.